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Ascending limb of Henle loop **Prostaglandins** **Magnesium**
Free water clearance **Bartter's syndrome** **Chloride reabsorption**

Baehler RW, Work J, Kotchen TA, McMorrow G, Guthrie G: Studies on the pathogenesis of Bartter's syndrome. Am J Med 1980; 69: 933–938.

Free water clearance and distal fractional chloride reabsorption were abnormally low in this patient with Bartter's syndrome. This series of investigations in a patient with Bartter's syndrome and hypomagnesemia was undertaken to determine if the defect in chloride transport in the ascending limb and the associated renal potassium wasting was specifically related to potassium depletion, increased prostaglandin production or magnesium depletion. Potassium repletion, indomethacin administration or magnesium repletion had no effect on the defect in free water clearance or in distal fractional chloride reabsorption. However, magnesium infusion eliminated renal potassium wasting suggesting that the proximate cause of Bartter's syndrome in this patient is a primary defect in the reabsorption of sodium chloride in the ascending limb and not renal potassium wasting.

Thiazide diuretic **Hydrochlorothiazide** **Triamterene**
Drug reaction **Renal failure** **Acute interstitial nephritis**

Magil AB, Ballon HS, Cameron EC, Rae A: Acute interstitial nephritis associated with thiazide diuretics. Clinical and pathologic observations in three cases. Am J Med 1980; 69: 939–943.

Renal failure insidiously developed in three patients treated with a combination of hydrochlorothiazide and triamterene. Two had eosinophilia and two had fever. Renal biopsy disclosed acute interstitial nephritis in all cases. Withdrawal of the thiazide resulted in resolution of the disease with renal function returning to normal in two patients and to near normal in the third. The sequence of events and histologic findings strongly suggest a drug-induced hypersensitivity reaction as the mechanism for the renal lesion. Although hydrochlorothiazide appears to be the drug mainly responsible for the reaction, it is suggested that triamterene may have a potentiating role.

Glomerulonephritis **Obstructive uropathy** **Immune complexes**
Antibody elution **Kidney disease** **Tubular epithelium**

Pascal RR, Sian CS, Brensilver JM, Kahn M, Lefavour GS: Demonstration of an antibody to tubular epithelium in glomerulonephritis associated with obstructive uropathy. Am J Med 1981; 69: 944–948.

In order to test the hypothesis that an immune complex glomerulonephritis can result from obstructive uropathy, we examined bilateral renal biopsy specimens from a patient with a unilateral ureterocele and nephrotic syndrome. In addition to immunofluorescent, light and electron microscopic evidence of bilateral glomerulonephritis, we were able to elute an antibody from each renal biopsy specimen which reacted specifically with renal tubular epithelium (RTE). We concluded that tubular injury results in an immune response to RTE-antigen and that subsequent deposition of RTE-antigen-antibody complexes in glomeruli produces a glomerulopathy with proteinuria.

Tubular basement membrane antibody **Interstitial nephritis**
Systemic lupus erythematosus **Renal disease**

Makker SP: Tubular basement membrane antibody-induced interstitial nephritis in systemic lupus erythematosus. Am J Med 1980; 69: 949–952

Interstitial nephritis characterized by linear staining for human IgG along the tubular basement membrane of proximal renal tubules is described in a child with systemic lupus erythematosus. Autoantibodies reacting with the tubular basement membrane of the proximal renal tubules and Bowman capsule of glomeruli were present in serum. The autoantibodies were specific and could be absorbed with a normal human renal cortical fraction. The findings suggest that in addition to the well-known, immune complex-mediated injury, autoantibody-induced interstitial nephritis can also rarely occur in systemic lupus erythematosus.

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